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10/735,439	12/11/2003	Jacob Bar-Tana	1567/70937-ZA /JPW/AG	2054

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EXAMINER	
ROYDS, LESLIE A	
ART UNIT	PAPER NUMBER
1614	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	02/23/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No.	Applicant(s)	
	10/735,439	BAR-TANA, JACOB	
	Examiner	Art Unit	
	Leslie A. Royds	1614	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *24 November 2006*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *29-54* is/are pending in the application.

4a) Of the above claim(s) *34,35,40,41,47,48,53 and 54* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *29-33,36-39,42-46 and 49-52* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *11 December 2003*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Claims 29-54 are presented for examination.

Applicant's Amendment and Exhibits A-G filed November 24, 2006 have each been received and entered into the present application.

Claims 29-54 are pending. Claims 34-35, 40-41, 47-48 and 53-54 are withdrawn from consideration pursuant to 37 C.F.R. 1.142(b). Claims 29-33, 36-39, 42-46 and 49-52 are pending and under examination. No claims have been amended, cancelled or newly added.

Applicant's submission of the Limatta et al. reference cited at page 5 of the Information Disclosure Statement (IDS) of December 11, 2003 has been received and entered into the present application. As reflected by the attached copy of form PTO-1449, the Examiner has considered the cited reference.

Applicant's arguments, filed November 24, 2006, have been fully considered but they are not deemed to be persuasive. Rejections and objections not reiterated from previous Office Actions are hereby withdrawn. The following rejections are either reiterated or newly applied. They constitute the complete set of rejections presently being applied to the instant application.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 29-33, 36-39 and 42-46 remain rejected under 35 U.S.C. 103(a) as being unpatentable over Bar-Tana ("Long Chain Dicarboxylic Acids: Hypolipidaemic, Antiobesity and Antidiabetic Activity", New Antidiabetic Drugs, 1990; cited by Applicant) in view of Hertz et al. ("Mode of Action of

Peroxisome Proliferators as Hypolipidemic Drugs", *Journal of Biological Chemistry*, 1995; cited by Applicant) and Ferrannini et al. ("Hyperinsulinaemia: The Key Features of a Cardiovascular and Metabolic Syndrome", *Diabetologia*, 1991), each already of record, for the reasons of record set forth at pages 7-11 of the previous Office Action dated June 20, 2006, of which said reasons are herein incorporated by reference.

Present claims 49-52 are withdrawn from the present rejection because the reference to Bar-Tana states that the cholesterol content of HDL remained essentially unaffected in the normal rat and that the 70-90% decrease in plasma cholesterol in the nephrotic rat was in plasma nonHDL-cholesterol. Please see the paragraph bridging pages 158-159 of Bar-Tana. Accordingly, this is an insufficient teaching to render the presently claimed method to increasing plasma levels of HDL cholesterol (see, e.g., claims 49-52) obvious in view of Bar-Tana.

Applicant traverses the present rejection, stating that Bar-Tana is an academic study of M16 in normal and nephrotic rats, not in humans, and the one of ordinary skill in the art would not have expected to results shown in the rats to be predictive of the results in humans. Applicant further argues that Hertz et al. does not remedy the deficiencies of Bar-Tana and that the failure of a related compound, C1-DICA, to have hypolipidemic effects in humans despite its efficacy in rodents and the failure of M16 to exhibit any hypotriglyceridemia effect in hamsters is clearly indicative of the fact that rats cannot serve as predictive models for the same activity in humans. Further, Applicant states that claim 29 is directed to the treatment of Syndrome X, which comprises combined hypertriglyceridemia/hypercholesterolemia/low HDL-cholesterol, i.e., characterized by the presence of all three of said conditions, and there is no reason to assume that a medicament that has activity in alleviating one or two of the symptoms would be relevant to the treatment of a syndrome containing all three conditions.

Applicant's traversal has been fully and carefully considered in its entirety, but fails to be persuasive.

First, Applicant's alleges that the study of M16 in normal and nephrotic rats is not predictive of the results in humans for the following reasons:

(1) rat lipoprotein profiles and metabolism do not simulate the human case;

(2) nephrotic dyslipoproteinemia in rats has nothing to do with the human disease and, therefore, cannot serve as a model for dyslipoproteinemic human patients;

(3) the apparent decrease in HDL-cholesterol percentage in the nephrotic rat just reflects the robust increase in VLDL and LDL cholesterol and, in fact, the absolute concentration of HDL cholesterol is massively increased by PAN nephrosis in the rat (relying on Bar-Tana et al. at Tables 1 and 3), which directly contrasts the low HDL cholesterol in human dyslipoproteinemia; and

(4) the effect of M16 on HDL cholesterol in the normolipemic or nephrotic rat is contrary to, and could not predict, its therapeutic effect in humans because M16 treatment in normal rats lowered HDL levels and had no change on HDL levels in nephrotic rats (relying again on Bar-Tana et al. at Tables 1 and 3), which contrasts the increase in HDL-cholesterol in human dyslipoproteinemic patients as seen in Applicant's clinical trials.

Regarding Applicant's statements raised *supra* as points (1) and (2), Applicant alleges throughout the remarks that the rat, either normal or nephrotic induced via PAN (puromycin aminonucleoside), cannot serve as an animal model predictive of the same efficacy in human patients because rat lipoprotein profiles and metabolism do not simulate the human case and the nephrotic dyslipoproteinemia in rats has nothing to do with human disease, but provides no evidence in support of the allegation that rats are not art-accepted models predictive of efficacy in humans. Accordingly, such remarks are no more than allegations without factual support. Please see, e.g., MPEP §716.01(c)[R-2](II), which states, "The arguments of counsel cannot take the place of evidence in the record. *In re Schulze*, 346 F.2d 600, 602, 145 USPQ 716, 718 (CCPA 1965)."

In fact, Bar-Tana expressly states to the contrary, teaching that the efficacy demonstrated by M16 is clearly suggestive of adapting the tested therapy for use *in vivo* in humans. Please reference Bar-Tana at the abstract, which states, "The overall pharmacological effect in rodents appears to point to the potential use of these drugs in the treatment of hyperlipaemic-obese-diabetic syndromes." (page 157)

Regarding Applicant's statements raised *supra* as points (3) and (4), Applicant relies upon the teachings of "Bar-Tana et al." at Tables 1 and 3, but does not clearly set forth to what reference he refers. Bar-Tana as cited by the Examiner contains no such tables, so it is obvious that Applicant is referring to extrinsic evidence, but has not clearly or deliberately identified to what document he refers. Accordingly, this evidence cannot be afforded the significance as Applicant has urged because the Examiner has not been adequately informed of the documents upon which Applicant's rely and, therefore, cannot properly considered the totality of the evidence as a whole.

It is further noted that, upon reconsideration of the teachings of Bar-Tana, that the rejection of present claims 49-52 has been withdrawn because Bar-Tana states that the cholesterol content of HDL remained essentially unaffected in the normal rat and that the 70-90% decrease in plasma cholesterol in the nephrotic rat was in plasma nonHDL-cholesterol. Please see the paragraph bridging pages 158-159 of Bar-Tana. Accordingly, this is an insufficient teaching to render the presently claimed method to increasing plasma levels of HDL cholesterol (see, e.g., claims 49-52) obvious in view of Bar-Tana.

Applicant provides extensive discussion regarding the two pathways by via HNF-4alpha-responsive genes are suppressed, i.e., an indirect pathway via the activation of PPARalpha/RXRalpha by free PP acid and the direct pathway that requires the respective CoA thioesters (PP-CoA) and results in the inhibition of HNF-4alpha by PP-CoA independently of PPARalpha as presented in Hertz et al. However, Applicant is considering the reference not as it was combined with Bar-Tana, but rather individually in the absence of the other references with which it was combined. Applicant is reminded that the rejections made under 35 U.S.C. 103(a) are based upon the combination of references and that the

references are relied upon in combination and are not meant to be considered separately as in a vacuum. It is the combination of all of the cited and relied upon references that make up the state of the art with regard to the claimed invention. Applicant's claimed invention fails to patentably distinguish over the state of the art represented by the combination of the cited references. Please reference *In re Young*, 403 F.2d 754, 159 USPQ 725 (CCPA 1968) and *In re Keller*, 642 F.2d 413, 208 USPQ 871 (CCPA 1981).

Moreover, rejections under 35 U.S.C. 103(a) are based upon combinations of references, where the secondary references are cited to reconcile the deficiencies of the primary reference with the knowledge generally available to one of ordinary skill in the art to show that the differences between Applicant's invention and the prior art are such that they would have been modifications that were *prima facie* obvious to the skilled artisan. It is noted that the claimed invention is not required to be expressly suggested in its entirety by any one or all of the references cited under 35 U.S.C. 103(a). Rather the test in what the combined teachings of the references would have suggested to those of ordinary skill in the art. Please see also *In re Keller*, 642 F.2d 413, 208 USPQ 871 (CCPA 1981).

In the instant case, Hertz et al. was relied upon solely for its teaching that M16 was known in the art to have efficacy in treating hypertriglyceridemia or combined hypertriglyceridemia and hypercholesterolemia (see, e.g., p.13470, col.1 of Hertz et al.) and, therefore, provides the reasonable expectation of success that a compound with efficacy in treating combined hypertriglyceridemia/hypercholesterolemia would also have efficacy in treating dyslipoproteinemia, which is known to be characterized by the concomitant presence of hypertriglyceridemia and hypercholesterolemia. Though Hertz et al. may also discuss different pathways of suppression HNF-4alpha-responsive genes, such disclosure does not constitute a teaching away from the broader teachings of the reference as a whole, namely the clear and express acknowledgement of the efficacy of M16 in treating both hypertriglyceridemia and hypercholesterolemia.

Further, Applicant's argument that a related compound, C1-DICA, had hypolipidemic effects in

humans despite its efficacy in rodents has been considered, but is not persuasive because it is directed to a chemically and structurally different compound and, therefore, would have been reasonably expected to have unique activity and unique therapeutic effects that cannot be reasonably extrapolated out to any other dicarboxylic acid, in the absence of any evidentiary basis to do so. In other words, the fact that C1-DICA may have efficacy in rodents but not humans provides absolutely no reason to doubt that the M16 studied in Bar-Tana would also not have efficacy in humans because it is a distinctly different compound to that claimed and is, therefore, not relevant in establishing the non-obviousness of the presently claimed methods of administering M16 to humans.

Applicant additionally argues that the failure of M16 to exhibit any hypotriglyceridemic effect in hamsters is also clearly indicative of the fact that rats cannot serve as predictive models of the same activity in humans. Applicant assumes that since hamsters are more closely related to humans in their lipoprotein profile than rats that one of skill in the art would not have been motivated to use M16 in humans based upon their inactivity in hamsters and relies upon various references in support of this position, but has provided none of these references in the record for consideration. Accordingly, and further in view of the fact that Bar-Tana does expressly suggest the adaptation of the disclosed therapy for use in humans as previously stated *supra*, Applicant's allegation that the skilled artisan would have had no motivation to use M16 in humans based upon its inactivity in hamsters is clearly not persuasive.

Lastly, regarding Applicant's assertion that Syndrome X (claim 29) comprises all three of the conditions hypertriglyceridemia, hypercholesterolemia and low HDL cholesterol and that there is no reason to assume that a medicament that has activity in alleviating one or two of the symptoms would be relevant to the treatment of a syndrome containing all three conditions, Applicant appears to have ignored the fact that he readily admitted on the record that Syndrome X comprises *some or all* of the three cited conditions and that the treatment of any one or more would have also treated Syndrome X. Please reference the remarks at page 7 of the response filed April 11, 2006, which states, "***As disclosed in the***

instant specification, this invention relates to novel methods of treating Syndrome X, which comprises some or all of dyslipoproteinemia (which itself manifests hypercholesterolemia-hypertriglyceridemia, and low HDL-cholesterol), obesity, impaired glucose tolerance, essential hypertension and thrombogenic/fibrinolytic defects (see, for example, page 10, last full paragraph, of the subject specification). Therefore, successful treatment of any of these conditions would results in improvement of Syndrome X."

As evidenced by the teachings of the references cited in the instant rejection, Bar-Tana in view of Hertz et al. and Ferrannini et al. clearly teaches the efficacy of M16 in, *at the very least,* one of the claimed conditions that comprises Sydrome X. Accordingly, and as per Applicant's admission on the record, the fact that M16 has activity in treating any of the conditions of dyslipoproteinemia, which itself is comprised of hypercholesterolemia, hypertriglyceridemia, and low HDL-cholesterol, would result in improvement of Syndrome X, absent factual evidence to the contrary. Therefore, Applicant's present allegation that the fact that the references do not teach all three conditions and, thus, do not teach Syndrome X is irrelevant, because they teach at least one of the conditions, which Applicant has stated on the record is sufficient to treat Syndrome X.

For these reasons, and those previously made of record at pages 7-11 of the Office Action dated June 20, 2006, of which said reasons are herein incorporated by reference, rejection of claims 29-33, 36-39 and 42-46 remains proper and is **maintained**.

Double Patenting

Obviousness-Type Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226

(Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Provisional Rejection

The provisional rejection of claims 36-39, 42-46 and 49-52 under the judicially created doctrine of obviousness-type double patenting over claims 29-34 of U.S. Patent Application No. 10/735,452 is hereby **withdrawn** because the '452 is no longer pending and has been abandoned.

Non-Provisional Rejections

Claims 36-39, 42-46 and 49-52 remain rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 5 and 19 of U.S. Patent No. 4,689,344 in view of Bar-Tana ("Long-Chain Dicarboxylic Acids: Hypolipiaemic, Antiobesity and Antidiabetic Activity", New Antidiabetic Drugs, 1990; cited by Applicant), each already of record, for the reasons of record set forth at pages 13-14 of the previous Office Action dated June 20, 2006, of which said reasons are herein incorporated by reference.

Applicant traverses the present rejection, stating that the present claims are directed to the treatment of humans and that Bar-Tana describes the treatment of nephrotic rats and the patent describes experiments in rats. Applicant further states that patented claim 5 is improperly included in the rejection because it is directed to a pharmaceutical composition and not a methods, i.e., the same statutory category of invention. Applicant also states that the patented claims recite the reduction of serum cholesterol and not the same therapeutic objectives recited in present claims 36, 42 or 49.

Applicant's traversal has been fully and carefully considered in its entirety, but fails to be persuasive.

First, Applicant's argument that the present claims are directed to the treatment of humans and Bar-Tana teaches the treatment of nephrotic rats and that the patent describes experiments in rats is not persuasive. Both the present claims and the patented claims are each directed to the administration of the identical M16 compound to a *human subject*. It is irrelevant that the patent describes experiments in rats because the claims expressly recite the use of the same M16 compound in humans. Regarding the fact that Bar-Tana teaches the treatment of nephrotic rats, Bar-Tana was cited for its teaching of the hypotriglyceridemic and hypocholesterolemic properties of M16 that were known in the art at the time of the invention. Regardless of the fact that Bar-Tana teaches its use in nephrotic rats, it remains that the patented claims recite the administration of the identical M16 compound to an identical host, i.e., a human subject, as presently claimed and, therefore, even in the absence of the teachings of Bar-Tana, the claimed effects on the treatment of dyslipoproteinemia, plasma triglycerides or plasma HDL cholesterol are all considered necessarily present in the method disclosed by the patent because products of identical composition cannot have mutually exclusive properties when administered under the same circumstances, i.e., in a human host. Accordingly, whatever other properties or therapeutic benefits Applicant has now attributed to the M16 compound (i.e., the claimed objectives of treating dyslipoproteinemia, lowering plasma triglycerides or increasing HDL cholesterol) are necessarily present in the patented claims, whether recognized by the patentee at the time of the invention or not, because the same compound (i.e., M16) is being administered to the same host (i.e., a human subject). Properties or effects of a compound are not severable from the compound itself when administered under identical conditions.

Second, patented claim 5 is purposefully included in the rejection because patented claim 19 is directly dependent upon claim 5 to define the compounds to be administered in the method of claim 19. Accordingly, the claim is solely relied upon for its teaching of the compounds for administration in the

method for reducing serum cholesterol as claimed in present claim 19 and is not an improper double patenting rejection based upon different statutory categories of invention.

For these reasons, and those made of record at pages 13-14 of the previous Office Action dated June 20, 2006, rejection of claims 36-39, 42-46 and 49-52 remains proper and is **maintained**.

Claims 29-33 remain rejected under the judicially created doctrine of obviousness-type double patenting over claims 1-3 and 8 of U.S. Patent No. 6,303,653, already of record, for the reasons of record set forth at pages 14-15 of the previous Office Action dated June 20, 2006, of which said reasons are herein incorporated by reference.

Applicant traverses the present rejection, stating that Syndrome X characteristics mediated by HNF-4alpha only comprise a partial group of the Syndrome X phenotype and that merely modulating HNF-4alpha activity as claimed in the patent may result in incomplete treatment, where Applicant's claimed method offers a comprehensive treatment mode that surpasses the performance of methods limited to modulating HNF-4alpha activity alone.

Applicant's traversal has been fully and carefully considered in its entirety, but fails to be persuasive.

Patented claims 1-3 and 8 clearly and unequivocally provide for the treatment of Syndrome X *per se* (see, e.g., patented claim 8). Accordingly, an attempt to show a patentable distinction over the patented claims by stating that the patented claims provide for the incomplete treatment of Syndrome X is clearly not persuasive because the patented claims clearly recite "Syndrome X". Furthermore, the rejection was set forth insofar as the present claims anticipate the patented claims. In other words, the fact that Applicant has now admitted on the record that the presently claimed method is a comprehensive treatment mode that surpasses the incomplete method of the patent clearly supports the conclusion that the present claims anticipate the patented claims because a method that encompasses all treatment aspects

of Syndrome X (as in the present claims) would clearly anticipate a subset of treatment aspects of Syndrome X (as in the patented claims).

For these reasons, and those made of record at pages 14-15 of the previous Office Action dated June 20, 2006, rejection of claims 29-32 remains proper and is **maintained**.

Conclusion

Rejection of claims 29-33, 36-39, 42-46 and 49-52 remains proper and is **maintained**.

Claims 34-35, 40-41, 47-48 and 53-54 remain **withdrawn** from consideration pursuant to 37 C.F.R. 1.142(b).

No claims of the present application are allowed.

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire **THREE MONTHS** from the mailing date of this action. In the event a first reply is filed within **TWO MONTHS** of the mailing date of this final action and the advisory action is not mailed until after the end of the **THREE-MONTH** shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than **SIX MONTHS** from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Leslie A. Royds whose telephone number is (571)-272-6096. The examiner can normally be reached on Monday-Friday (9:00 AM-5:30 PM).

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ardin H. Marschel can be reached on (571)-272-0718. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Leslie A. Royds
Patent Examiner
Art Unit 1614

February 18, 2007

ARDIN H. MARSCHEL
SUPERVISORY PATENT EXAMINER